Letters to the editor [3] (multiple letters)

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Letters to the Editor

To the Editor:

Concerning the study conducted by Sapir and Gorup,[12] it is stated that one of the objectives is to assess the effect of monetary gain on the treatment of zygapophysial joint pain in cervical whiplash. This is a difficult objective indeed because the authors cannot isolate "monetary gain" as a confounding variable in the populations they study. Indeed, the point of such study seems moot.

Insurance fraud exists. Of that there is no question, and physicians have even been caught with their hand in the pie as well.[1] At the same time, even the more cynical of authors and researchers in this field think that this is an uncommon problem, and one does not perceive the need to conduct research to pummel those who lack this insight. I have not seen any publication declare a 100% malingering rate, or even 50%. One study of an already select group suggests a value of 25% of subjects showing evidence of lack of honest effort, but even then the authors do not presume this to equate with a lack of honest symptoms and suffering.[13] We can all agree that frank malingering, although it occurs, does not explain most subjects. Indeed, current models of whiplash are based on the recognition of a low rate of malingering and are built on the recognition that there are so many cultural factors engendering chronic pain that patients can be genuinely convinced they have chronic damage in their neck. As has been said before, for most human beings the capacity for self-deception is so great that there is no need for malingering.[8] If the purpose of the study by Sapir and Gorup[12] is to examine the hypothesis that either the prevalence of malingering (for presumably secondary gain) is low among litigants, or at least no higher than in nonlitigants, the following assumptions must be made for the methodology used by Sapir and Gorup[12] to reasonably address the research question:

1. That the prevalence of malingering is high among litigants; otherwise, if one assumes a 10–15% rate of malingering, a power analysis shows that a much larger number of subjects is required to ensure at least an 80% chance of not failing to detect a real difference in the populations studied.

2. That malingering for secondary gain means malingering for money; that is, assuming that secondary gain refers to money and mainly money. Actually, Fishbain[6] and Ferrari and Kwan[2,3] have reviewed the literature on secondary gain and consider that there are at least 13 possible types of secondary gain, and money is just one of them. Because monetary compensation is the only difference between nonlitigants and litigants, there are still 12 other forms of secondary gain available to both groups.

3. That those who pursue secondary gain do so through malingering only. Instead, current understanding of cognitive theory suggests that the interaction of secondary gain and adoption of the sick role may involve preconscious motivations[2,3] as readily as they do conscious motivations.

4. That nonlitigants do not malinger. How valid an assumption is that when they have many forms of secondary gain available to them? Do nonlitigants ever have access to wage supplements while they rest on their laurels at home? Do nonlitigants not desire the other 12 forms of secondary gain?

5. That a malingerer never responds to therapy. The authors consider this, as it would indeed be sensible as a malingerer, to respond to a therapy that "proves" you have a serious problem. This is their big chance to prove they are "for real." The malingerer remarks, "See, I have this problem deep in my neck and they proved it by injecting a drug there. I must be telling the truth.

"Of course, I am not cured, not until I speak with my lawyer." A wise malingerer would respond to therapy somewhat or at least for some period of time, with it to recur eventually and confirm that even "these wisest of doctors cannot cure me." The authors make various assumptions about exactly when and how a malingerer would decide to respond to therapy, assumptions that they cannot readily test.

This study deals with a difficult research question, difficult because really the only ones who know the answer are not telling. But at the least the researcher has to make a more dramatic effort to answer the question. The current effort is more based on assumptions (a great many) than on a valid research approach. This is not surprising, for perhaps it is as Bertrand Russell once wrote[11]:

> The method of 'postulating' what we want has many advantages; they are the same as the advantages of theft over honest toil.

Finally, Sapir and Gorup[12] have not demonstrated a facet joint arthropathy. What they have demonstrated is that if you insert a needle into someone's neck, some people then report less pain. Even Wallis et al.[14] themselves have stated that the procedure does not describe

what the pathology is, just that whatever structure is supplied by a certain nerve branch is no longer causing pain. Sapir and Gorup also state that the facet joint has been proven to be the cause of pain after whiplash. This is not true because the studies to which they refer do not allow for that conclusion at all.[4,5]

A final concern: Although it has not been investigated as much as it should be, postural abnormalities have been associated with chronic pain. Postural abnormalities, when burdened on healthy subjects, give them pain.[7,9,10,15,16] One could study the hypothesis that postural abnormalities, which must have some physical mechanism of inducing pain, do so through mechanical forces on the facet joint and related structures. There is research currently underway to investigate this particular aspect. Are we treating postural abnormalities with neurotomy instead of advice to return to normal activities despite pain, a lumbar roll, neck retractions, and back extension exercises?

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In Response:

We have reviewed Dr. Ferrari's letter, and we thank him for his interesting if unconventional response. His method of critiquing our article was to enumerate several assumptions that he asserted were essential for our methodology to "reasonably address the research question" that he imposed on our study, these being distinct from the actual question posed in our article.

Ferrari's method is, in actuality, a thinly disguised logical fallacy in which he shoehorned our approach into his own paradigm of whiplash injury and malingering, thus allowing him to define concepts that we did not raise in our article. We will not address criticisms of assumptions and tangent views that we did not make.

Logical fallacies aside, Ferrari needlessly complicated the methods of our study, which were simply to compare the response to cervical facet neurotomy of litigants to nonlitigants. Ferrari claimed that our methods were "more based on assumptions (a great many) than a valid research approach." What is there about a prospective controlled and blinded study that is not a valid research approach?

Although our results do not allow for the conclusion that all questions regarding whiplash and chronic pain have been answered, they contradict Ferrari's hypothesis that chronic pain after whiplash is a result of self-deception on the patient's part fostered by lawyers and doctors and, therefore, all that is needed is for the patient to be reassured that there is nothing wrong.

A review of Ferrari's citations after his letter reveals four of his own publications, all opinion pieces espousing similar concepts as those in the letter, using a similar approach. His approach applies nihilistic criteria to research, which he eschews. At the same time, he improperly extrapolates the results of anecdotal and methodologically flawed research to fit his ideas about chronic pain after whiplash while ignoring the valid and vigorously conducted body of research supporting the pathophysiologic process of whiplash injury.

No research can withstand the cynical scrutiny that Ferrari has leveled at our article. In fact, his own arguments become a tautology in that they cannot stand up to his own methods of scrutiny. We cannot allow his views to thwart us or other investigators from continuing proper and legitimate scientific inquiry. We regretfully concede that it is unlikely that any publication will ever convince Ferrari and others who espouse his viewpoint that whiplash injuries can and do result in chronic pain.

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To the Editor:

I would like to comment on an article by Leclaire et al.[4] I disagree with the conclusions that were reached by the authors as well as by Dr. Deyo in the Point of View[2] that followed this article. First, choosing patients for inclusion into the study based on successful pain relief by one intra-articular zygapophysial joint injection clearly

bucks current diagnostic standards for the determination of zygapophysial joint-mediated pain. Schwarzer et al.[5] clearly showed in a controlled study that a single diagnostic zygapophysial joint block carries a false-positive rate of 38%. Leclaire et al.[4] appropriately indicated in their discussion that diagnosing zygapophysial joint-mediated pain is more effectively done *via* comparative anesthetic (and saline placebo) blocks. Yet to draw conclusion for the efficacy of lumbar radiofrequency zygapophysial joint denervation based on their inclusion criteria is not only misleading but fallacious. Dr. Deyo indicated that such a diagnostic regimen may be a very "exacting and demanding process," therefore of questionable use for widespread practice.[2] The current standard of diagnosing zygapophysial joint-mediated pain *via* comparative local anesthetic blocks is exacting, yet it is a standard that we must uphold for the sake of our patients and the proper diagnosis of their pain generator.

The method for radiofrequency neurotomy used in this study is also of question. If (as stated) the authors used the technique as modified from Shealy,[6] it was indeed an inappropriate technique to use. The method as described by Shealy[6] places the radiofrequency electrode in a relative perpendicular position to the nerve. Bogduk et al.[1] have clearly shown that the position of the radiofrequency electrode must be parallel to the nerve to create an optimal lesion. The authors noted in their discussion that creating an ineffective lesion was possible because of inaccurate anatomic localization. To publish this article, with such an evident possibility, and to draw conclusion regarding the efficacy of the procedure, based on this, is disconcerting.

Dreyfuss et al.[3] have shown that, in a well-selected patient, radiofrequency neurotomy for lumbar zygapophysial joint pain is an efficacious treatment. The fact that Dr. Deyo[2] finds the present article an "important contribution," "sobering," and a "well-designed trial" shows not only a lack of understanding of patient selection for this procedure but also a lack of appreciation of the technical application of percutaneous radiofrequency neurotomy. Fortunately, the authors of the article do not categorically dismiss radiofrequency neurotomy as a therapeutic approach.

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In Response:

We respect the opinion of Dr. Diamant on our article. Our discussion on the results clearly put forward the possible limitations of the results of this study as also pointed out by Dr. Deyo. The authors do not dismiss radiofrequency neurotomy as a therapeutic approach. We look forward to larger randomized trials to better delineate the benefit and limitations of this therapeutic approach.

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To the Editor:

When I first reviewed the abstract for the Leclaire et al.[1] study, I was surprised and perplexed. The authors' conclusions drastically contradicted my experiences with neurotomies over the past few years. Reading the article was both a relief and a disappointment.

The relief was because the results could have been predicted from the Methods section alone. The study subjects were those who had experienced significant relief of their low back pain for at least 24 hours during the week after intra-articular facet injections. The medications injected were lidocaine hydrochloride and triamcinolone acetonide.

The first flaw in this selection procedure is the undefined "significant relief." Was this 25% relief or 100% relief? The difference is important because a patient with facet pain from two levels may have 50% relief from denervation of one level, yet the remaining pain may prevent discernible functional improvement. And that was the authors' measuring stick.

The second flaw is that the authors completely ignored the work of Schwarzer et al.,[3] who showed in 1994 that the false-positive rate from uncontrolled lumbar diagnostic facet nerve blocks was 38%. Assuming the 15–40% prevalence of lumbar facet pain found by Schwarzer et al.,[4,5] then we can estimate that only 10 to 18 of the 35 patients in the group that received radiofrequency neurotomies had lumbar facet–mediated pain to treat.

The third flaw is that the authors used lidocaine hydrochloride (without epinephrine) and triamcinolone acetonide for the facet joint blocks. Lidocaine is a short-acting anesthetic, with an average duration of action of 1–3 hours. Triamcinolone acetonide is a corticosteroid with a controversial potential to provide some relief for facet-mediated pain but no recognized value for diagnosis of a facet disorder and no expected benefit in the first 24 hours. Thus, the duration of relief required by the authors for study admission was inconsistent with the known and recognized effects of either of the medications used.

The fourth, and most damaging, flaw is that the authors defined the inclusion criteria to include only those patients who reported at least 24 hours of significant lumbar pain relief in the week after the facet joint blocks. This result is an exaggerated version of what Lord et al.[2] described as a "prolonged" response in their analysis of cervical confirmatory diagnostic blocks. They found that when a prolonged response occurred, the patient did not have facet-mediated pain >80% of the time.

In other words, Leclaire et al.[1] chose an inclusion criterion that practically eliminated any patients with lumbar facet pain from the study. Their failure to find benefit from facet neurotomies for patients who did not have facet-mediated pain is not a revelation, it's a truism. Next time they want to test the efficacy of a treatment, they should try it on patients who have the relevant disorder.

My disappointment was from the realization that some of the editors of *Spine* are authors of research that demonstrates the flaws of this article. It is beyond unfortunate that these editors were not consulted before this article was published.

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In Response:

The selection procedure for the treatment of patients in whom the low back pain is estimated to be caused by a facet joint problem is difficult. According to the common practice at the time of this study (the protocol was written in 1991 and the study began in 1993), we selected the best criterion available, *e.g.*, "subjects who had experienced significant relief of their low back pain for at least 24 hours during the week after the injection." The common practice was also to use a local anesthetic and cortisone for the treatment of low back pain with a presumed facet etiology. The study of Schwarzer et al.[2] on the false-positive rate in uncontrolled lumbar diagnostic facet blocks reported by Ketroser was published in 1994, 1 year after the beginning of our study.

There is no evidence that setting a percentage of relief as a selection criterion has proven to be more valid in assessing the success of a lumbar facet block than the opinions of the patient and the attending physician on a "significant" relief, which was our first inclusion criterion.

Ketroser reports the study of Lord et al.[1] published in 1995. This study addresses cervical facet joint. The conclusions of their study on the response to diagnostic blocks cannot be applied to lumbar facet-mediated pain. They should be validated by a similar methodology at the lumbar level.

We strongly disagree with Ketroser's statement that the selection procedure "practically eliminated any patients with lumbar facet pain from the study." First, despite their limits, the selection criteria identified a majority of patients with a lumbar facet pain, even when looking at the results of the Schwarzer et al. study.[2] Second, the results at 4 weeks showed that there was a statistically significant relief in the Roland-Morris score in the neurotomy group compared with the placebo group, improvement that did not persist at 12 weeks. It is doubtful that such a response would have been seen in patients with no lumbar facet problem.

We are aware of the limitations of this study, which were outlined in our discussion. Despite one positive study (with a small sample size) published in 1999,[3] we regard the lumbar radiofrequency facet denervation as an unproven therapy, as stated by Deyo in his Point of View on our study. There is definitely a need for large and rigorously designated studies to assess the benefits of this treatment procedure for low back pain.

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Luc Fortin, MD, MS

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To the Editor:

In a recent article in this journal, Puniello et al.[6] addressed lifting styles in elderly subjects. They developed a classification of the lifting styles spontaneously adopted by the subjects and showed the style to be related to strength capacity of hip and knee extensor muscles. Furthermore, they addressed the effects lifting style has on stability. In view of the high risk related to falls during lifting and the balance problem elderly subjects often incur, we acknowledge the importance of this topic. However, we submit that the conclusions and practical advice formulated by the authors are premature. The authors conclude that a leg lifting style should be advised to the elderly because of the higher stability and lower

spinal compression imposed. The first presumed benefit of leg lifting is based on data presented in their article. The second is based on a previous publication.[5] In his commentary on the article, Dr. Marras stresses that the question (which lifting style is to be preferred) involves weighting of more aspects than those covered by the authors. We hold that even those aspects considered in the study by Puniello et al.[6] do not warrant the conclusion drawn.

Puniello et al.[6] gauge stability in lifting from the horizontal distance between the center of pressure of the ground reaction force and the center of mass of the subject plus load. They claim this distance to be the moment arm of the ground reaction force with respect to the center of mass. The rationale for interpreting this in terms of stability is based on the idea that the moment caused by the ground reaction force is disturbing balance by causing rotation about the center of mass. There are several objections to both this line of reasoning and the operationalization used. To start with the latter, the horizontal distance only reflects the moment arm when the ground reaction force is vertical. Not only is this not the case in lifting, the direction of the ground reaction also is dependent on the lifting style used.[7] Second, the moment arm and the magnitudes of the ground reaction force together determine the magnitude of the moment. Because the ground reaction force is substantially higher in leg lifting,[7] the moment arm alone does not allow conclusions to be drawn with respect to the moment magnitude from an indication of the moment arm alone. Of a more fundamental nature is the question of whether the moment adequately reflects the threat to stability. It should be realized that the moment of the ground reaction force is a necessary consequence of the movements performed by the subject. No movement would be possible without creating such a moment. Of course, it can be stated that any movement is a threat to stability and that, as such, movements involving a higher angular momentum impose a larger threat to balance. However, stability is not only determined by the rotational movement about the center of mass reflected in this moment, but also by the horizontal linear displacements of the center of mass reflected in the horizontal ground reaction force. When balance is perturbed during lifting, subjects need to correct both components.[3] Finally, the support surface during leg lifting is often reduced because subjects are unable to keep their heels in contact with ground, which compound the balance threat. In all our experiments in which perturbations during lifting were applied,[2,7] we found balance loss to be more common during leg lifting than back lifting. This finding was recently confirmed by work from another group.[1]

Finally, based on a study by Leskinen et al.,[5] the authors assume that leg lifting imposes lower compression loads on the spine than back lifting. An extensive review of the biomechanical literature on this topic has shown that there is no sufficient evidence to support this assumption. Most recent studies, which use more sophisticated models and carefully controlled experimental conditions, report higher instead of lower compression forces in leg lifting *versus* back lifting.[4]

In conclusion, we believe that the conclusions drawn by Puniello et al.[6] are insufficiently supported by the literature and their own data. Although we applaud their systematic classification of lifting styles and underline the importance of studying lifting in the elderly, we think that premature and possibly incorrect practical inferences from limited experimental data should be avoided.

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In Response:

We would like to thank Dr. van Dieën and Dr. Toussaint for their comments regarding our study. They raise valuable and interesting points, which facilitate debate and prompt further discussion, comparing results from different studies. We are pleased to be invited to participate in this discussion.

First, we think it is prudent to point out some experimental design and procedural differences between our study and those discussed by Drs. van Dieën and Toussaint. Our initial start position and lifting procedure were very different from that of Toussaint et al.[8] In their first experiment, a box was placed 0.3 cm in front of the toes, and subjects were instructed to perform a fast lifting movement, which was reinforced by a metronome. Subjects lifted the box to breast height and then lowered it to the floor. In their second experiment, subjects lifted a barbell, which "was placed in front of the toes at such a distance (heel-barbell 0.616 $\pm$ 0.054 m) that the subject was just able to pick it up (start distance in front of toes was 14% of body height)." The type of lift was specified,

and the trajectory of the barbell was directed to be in front of the body, guided by flexible wands. Subjects performed five to seven such trials, lifting the barbell to acromion height and then lowering it to the floor. This differs greatly from our study, in which the box was placed on the floor with the corners just in front of the second toes. Our subjects performed a freestyle lift at their preferred speed and placed the box on an umbilicus height table in front of them. These differences might account for the discrepancy of the ground reaction force direction between the studies. Our conclusions regarding postural stability of "frail" elders during lifting might be more valid because we tested their preferred lifting method, which they are most likely to use functionally.

With regard to comments about the center of mass − center of pressure difference, or moment arm, we defend this measure as an index of stability. The peak sagittal plane inclination (from vertical) of the ground force vector during the lift activity was only 1.4 ± 0.4° across subjects, and there was no significant difference in inclination among the lifting style groups ($P = 0.972$). Furthermore, there was no significant difference between groups in the maximum ground reaction force ($P = 0.219$). This supports our conclusion that the significantly greater maximum moment arm measured for the back dominant group does indeed suggest a greater potential for postural instability. Indeed, Drs. Dieën and Toussaint are correct when they point out that "the moment of the ground reaction force is a necessary consequence of the movements performed by the subject." Indeed, we have stated this in several prior publications.[4,6,7,10]

Drs. van Dieën and Toussaint also discussed postural stability with perturbed balance during lifting and reported back lift to be more stable than leg lift. In their studies balance was perturbed by unexpectedly altering the load of the box[8] and by increasing the speed of the lift until subjects lost their balance.[1] This also differs from our study because our subjects were told the constant weight of the load, and they also had one practice lifting trial before the two experimental trials. Perhaps most importantly, our subjects' heels were on the floor (feet flat on the floor) during the entire lift. The peak foot angle from horizontal (feet flat) across subjects during the lift activity was 2.7 ± 2.2°, and there was no significant difference among the lifting style groups ($P = 0.322$). This fact along with the difference in initial start position, and our subjects' using their preferred lifting speed and style, might account for the different conclusions regarding stability during lifting.

With regard to spinal compression, a number of studies report that leg lift decreases torque and spinal compression, when the load is positioned between the legs. If the load is held away from the body, there is no spinal compression difference between lifting styles, or there is reportedly higher spinal compression with leg lift.[3] Drs. van Dieën and Toussaint also reviewed a study in which there was lower moment in squat lift with a freestyle lift. Our subjects lifted with the box between the feet. We developed our power analysis for lift technique classification because the majority of our subjects used a combined back/leg lift with the vertical ground reaction force near the knee joint center, so we could not use the method described by Toussaint et al.[9] Therefore, we used the terms "back dominant" and "leg dominant strategy."

We acknowledge that our recommendations for lifting style may be too strongly worded. Our results may only be generalizable to the conditions simulated by the functional conditions of our experimental procedure; we recommend leg dominant strategy with the load held between the legs to promote safety for elderly subjects. DiFabbio wrote an editorial stating that two people could review the same body of literature and draw entirely different conclusions, based on their interpretation of the evidence.[2] We acknowledge that there are many factors that must be taken into account in recommending a lifting strategy.[5] We encourage critique of our work and recommend review of the specific study procedures to allow careful comparison of results of different studies. We would like to thank Drs. van Dieën and Toussaint for their letter, Dr. Maraas for his commentary, and the Editor for the opportunity to contribute to the multifaceted discussion of lifting strategy.

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To the Editor:

With keen interest I read the case report by Sengupta et al.[5] regarding a new technique for correction of an iatrogenic extension deformity in ankylosing spondylitis. The authors described the treatment of a patient with a severe thoracolumbar kyphotic deformity resulting from

ankylosing spondylitis in which a lordosing osteotomy of the thoracic spine caused an extension deformity of the cervical spine. The case illustrates well the need for adequate deformity planning for sagittal plane corrective osteotomies of the spine in ankylosing spondylitis.

As to be expected, extension osteotomies of the spine in ankylosing spondylitis alter both the chin-brow to vertical angle and the sagittal balance of the spine. The effect of a spinal osteotomy on the sagittal balance of the spine depends on both the correction angle and the level of osteotomy simultaneously. Also, the effect of a spinal osteotomy on the chin-brow to vertical angle is the same as the osteotomy angle, irrespective of the osteotomy level. Therefore, accurate preoperative planning, including the degree of correction required and the level to operate on, is essential for reliable prediction of the effect of such a surgical procedure. The authors based their preoperative assessment of the thoracolumbar kyphotic deformity on the measurement of the thoracic kyphosis, the (C2) plumbline, and the chin-brow to vertical angle. I would like to put forward some notes on the interactions between these three aspects of deformity planning.

First, the measured thoracic kyphosis in this patient was 120°. The reported patient underwent a closing wedge osteotomy at T6 and T8. A total osteotomy angle of 75° was achieved. This is a magnificent correction if we realize that thoracic correction is strongly limited by the rib cage. In addition, the relative narrow thoracic spinal canal renders the midthoracic spinal cord more vulnerable to perioperative injury than the cauda equina in its spacious spinal canal. For this reason the thoracolumbar kyphotic deformity is preferable, corrected by a lordosing osteotomy of the lumbar spine.[8] Furthermore, the overall correction is greatest when the intervention is performed at the lowest possible level of the lumbar spine.[6]

Second, sagittal spinal balance can be determined by a vertical plumbline, dropped from the most cephalad visible vertebra on standing lateral full-length radiographs of the spine. Several authors tried to quantify normal and abnormal sagittal spinal balance.[1–4] It should be noted, however, that these studies deal with subjects with normal spinal segmental mobility. Thus, disturbances of sagittal balance can be compensated for by segmental movements within the spine. Naturally, in patients with ankylosing spondylitis this is not possible; they rely on compensatory movements in the hip, knee, and ankle joints. Usually, patients extend their hips and flex their knees to prevent themselves from falling forward. By neglecting the position of the lower extremities, the accuracy of reporting sagittal plane deformity may be questionable.[7]

Third, the severity of the thoracolumbar kyphotic deformity can also be assessed by the chin-brow to vertical angle. This case illustrates the importance of functional restoration of the chin-brow to vertical angle. In addition, surgical correction of the spine based on the correction of the chin-brow to vertical angle alone would restore the normal view angle but leaves the hip joints in full extension.

Consequently, assessment of the thoracolumbar kyphotic deformity has to be performed in such a way that the joint position of the lower extremities is considered. Therefore, we recently developed a biomechanical method for deformity planning for sagittal plane corrective osteotomies of the spine in ankylosing spondylitis that renders measurements of the C7 plumbline independent of any compensatory position of the lower extremities.[9] Then, deformity planning for lumbar osteotomy in ankylosing spondylitis can be performed with the use of a mathematical analysis with trigonometric equations. Because restoration of sagittal spinal balance relies on precise deformity planning, this method is justified to prevent overcorrection of the visual field upward as described in the case.

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In Response:

We appreciate the opportunity to respond to Dr. Van Royen's comments on our article. Dr. Van Royen has generally agreed to the need for adequate planning before corrective osteotomies for sagittal plane deformity of the spine in ankylosing spondylitis. I would like to clarify certain points of misunderstanding in our article.

We agree that the upper lumbar spine is the preferred site for osteotomy to correct a kyphotic deformity in ankylosing spondylitis. Our patient had a previous lumbar osteotomy at L3. Subsequent osteotomy was needed

for a recurrence of kyphotic deformity 10 years later. This was performed at the thoracic level to avoid dissection through the previous osteotomy site and also to place the osteotomy at the apex of the deformity. Because thoracic osteotomy achieves a lesser degree of kyphosis correction compared with a lumbar osteotomy, a two-level osteotomy had to be performed at T6 and T8.

Dr. Van Royen's statement that "overall correction is greatest when the intervention is performed at the lowest possible level" needs further clarification. The correction of the visual angle remains the same as the osteotomy angle, irrespective of the level of the osteotomy. In contrast, the posterior shift of the plumb line becomes greater the lower the level of osteotomy.

We do not agree with the statement that "the severity of the thoracolumbar kyphotic deformity can also be assessed by the chin-brow to vertical angle." The forward shift of the plumb line and the chin-brow to vertical angle are two independent variables. Patients may have a small chin-brow to vertical angle, with a large forward shift of the plumb line, when the cervical spine is ankylosed in relative extension or neutral position. The level of osteotomy has a divergent effect on these two variables, which has been explained in more detail in our article.

We agree that in the presence of global kyphosis in ankylosis spondylitis, patients extend their hips and flex their knees, as much as possible, to correct the sagittal balance and to achieve a forward gaze. A measurement of chin-brow to vertical angle and forward shift of the plumb line from the lateral radiograph taken in this posture will underestimate both the parameters. The simple way to resolve this problem is to take the lateral radiograph when the patient stands with relaxed hips, without making an effort to correct the spinal balance or forward gaze.

Van Royen et al[1] have described a precise method for planning deformity correction that involves the use of mathematical analysis with trigonometric equations to construct a nomogram for individual patients. The authors admitted that the "optimum position of the postoperative sagittal vertical axis (SVA) and sacral endplate angle (SEA) is not known." These are essential parameters for construction of the nomogram. They used this method only in two cases retrospectively, which indicates its potential complexity for routine use in prospective planning of sagittal plane deformity correction.

The authors expressed the need for "a more exact and controllable surgical procedure" and special measurement device like "a customized mechanical or computer-assisted goniometer that can be placed onto the transpedicular screws adjacent to the closing-wedge osteotomy" during surgery.

The deformity correction with present day surgical technique is a close approximation rather than an accurate reproduction of the calculated angle on the operation table.[2,3] We feel that the planning of level osteotomy, based on chin-brow to vertical angle and forward shift of the plumb line, as described in our article, is a simple and more practical method.

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To the Editor:

I read with great interest the impressive article by Musgrave et al.[9] demonstrating an association between estrogen replacement therapy and back pain among postmenopausal women. Despite fewer fractures and greater bone mineral density, women using estrogen replacement therapy reported significantly more back pain compared with never-users. As the authors note, these findings are consistent with previous research on the association of hormone therapy and low back pain.[1,13] The authors also discuss plausible mechanisms for explicating this association, with an emphasis on hormonal effects on joint laxity.

I would like to add to the authors' scholarly discussion of potential mechanisms by suggesting that the association of hormone replacement therapy with back pain may be due to more general effects of estrogen on nociceptive processing. Several lines of evidence are relevant here. First, back pain is not the only form of clinical pain associated with exogenous hormone use. LeResche et al.[8] demonstrated that both oral contraceptive use and hormone replacement therapy were associated with increased risk of temporomandibular disorder. Moreover, they demonstrated a dose–response association between risk for temporomandibular disorder and the dose of estrogen consumed. Relatedly, Wise et al.[14] found that postmenopausal women on estrogen replacement seeking treatment for orofacial pain reported significantly more severe pain compared with facial pain patients not using hormones. In addition, oral contraceptive use has been associated with increased risk of carpal tunnel syndrome.[3] Thus, increased risk of developing several painful clinical conditions is associated with exogenous hormone use.

In addition to their effects on clinical pain responses, ovarian hormones have been shown to influence laboratory-based measures of pain perception. For example, pain sensitivity fluctuates across the human menstrual cycle, such that for most forms of painful stimulation,

higher pain thresholds and tolerances are observed during the follicular *versus* the periovulatory and luteal phases.[10] In a previous study examining menstrual cycle effects on pain perception, we found that regardless of cycle phase, higher estrogen levels were associated with increased pain in response to thermal stimuli.[5] Of more direct relevance to the current topic, we recently demonstrated that postmenopausal women taking hormone replacement therapy displayed lower thermal pain thresholds and tolerances than postmenopausal women not taking hormone replacement therapy and men, while these latter two groups did not differ from each other.[4] Thus, increased estrogen, either endogenous or exogenous, has been associated with enhanced sensitivity to experimentally induced pain.

There are multiple mechanisms whereby ovarian hormones can alter pain perception, and we have previously suggested that these effects can occur at each of three levels of the nociceptive processing system.[6] First, several lines of evidence suggest that estrogen may increase the excitability of peripheral afferents. Second, hormonal factors can modulate nociceptive processing at the level of the dorsal horn of the spinal cord through their influence on the central nervous system activity of multiple neuromodulators involved in spinal nociceptive processing, including substance P,[2,7] amino acids such as γ-aminobutyric acid and glutamate, and other neurotransmitters (*e.g.*, dopamine, serotonin, and norepinephrine).[11] Third, gonadal hormones influence brain systems involved in nociception. For example, a recent study demonstrated a negative correlation between circulating estradiol and μ-opioid receptor binding in certain brain regions in humans.[12]

The authors are to be commended for their ambitious research, and their results add to a growing body of evidence suggesting that estrogen may increase the risk of experiencing greater pain from multiple sources. While ovarian hormones have peripheral effects that may have specific relevance to the increased incidence of back pain, I believe it is also worthwhile to consider the more general effects of sex hormones on the pain processing system. In the end, this may provide a more parsimonious explanation for the varieties of pain that appear vulnerable to the effects of estrogen.

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To the Editor:

We read with great interest the randomized, controlled trial of Karppinen et al.[2] describing periradicular infiltration for sciatica. This study was randomized and double blinded; however, it had results that do not correlate with previous controlled trials.[3,4,6,8,9] Transforaminal epidural injections have been associated with controversy since their introduction.[5] Multiple aspects of the controversy concerning transforaminal epidural injections, we believe, once again resurfaced with the publication of the article by Karppinen et al.[2] This study brings out the controversy with regards to terminology of the procedure itself, the technique utilized, randomization, and the outcome results.

The terminology describing transforaminal epidural injections has varied from nerve root injections to selective nerve root blocks, selective nerve root sleeve injections, selective epidurals, selective spinal nerve blocks, selective ventral ramus blocks, and now periradicular infiltration. However, it has been stated that selective nerve root block is not an appropriate term for either the diagnostic or the therapeutic procedure. Thus, for diagnostic purposes, selective spinal nerve block or selective ventral ramus block, and for therapeutic purposes, transforaminal epidural injection have been considered appropriate.[5] However, periradicular infiltration appears to be a new terminology, which probably encompasses not only the spinal nerve block but also transforaminal epidural injection. Karppinen et al.[2] have used the term periradicular infiltration interchangeably with transforaminal epidural injection, even though the injection vol-

umes were highly inconsistent. They injected contrast of 0.5–1.0 mL for diagnostic purposes followed by a therapeutic injection of methylprednisolone 40 mg, bupivacaine, or isotonic sodium chloride solution in a volume of 2 mL for L4 or L5 blocks and 3 mL for S1, presumably based on anatomic differences. It was not clear from the publication if the methylprednisolone was mixed with the bupivacaine or was injected before or followed by the bupivacaine. If these were combined in a solution, the amount of the injectate remaining at the target site would be extremely low because only <0.5 mL of 2–3 mL may remain at the target site. If the methylprednisolone were injected before the injection of bupivacaine, the bupivacaine would have flushed all or most of the methylprednisolone into the interlaminar epidural space. Thus, once again, the methylprednisolone would be far removed from the site of pathology, providing insufficient target concentrations. We have seen 1–3 mL of contrast extending into four or five segmental levels.

The process of randomization was flawed in that patients were recruited from general practitioners on the basis that they were presumably suffering from sciatica. This would be the equivalent of recruitment by advertisement, which is certainly not the ideal situation, especially to compare them with the patient population in the practices of interventional pain physicians.

The findings of MRI classification of symptomatic discs was highly variable with a significant number of patients having either a normal disc or a bulge and most patients having a disc extrusion. Further, there are flaws based on the reporting pattern in which the percent of improvement was reported as a unit for the group rather than each individual patient. To interpret the statistics appropriately, the standard practice is to report the number of patients with $>50\%$ pain relief and the number of patients with improvement in various other parameters. Reduction in leg pain, back pain, disability, *etc.*, when not reported for individual patients, is extremely difficult to interpret. In addition, this was a single injection study; thus, it is extremely difficult to extrapolate the results of this study to the actual population suffering with chronic low back pain and treated in interventional pain medicine settings.

It is also extremely difficult to explain so many differences in outcomes based on randomization itself. Concato et al.[1] showed that well-designed observational studies (with either a cohort or case–control design) do not systematically overestimate the magnitude of the effects of treatments compared with randomized, controlled trials on the same topic. Further, Pocock and Elbourne[7] observed that, in a systematic review of the evidence on a therapeutic topic, one needs to take into account the quality of the evidence because in any study, either a randomized or observational bias may exist either in design or analysis.

All these factors considered, the Karppinen et al.[2] study may be summarized as concluding that interlaminar injection under fluoroscopic visualization provides short-term effect, a finding that is consistent with previous evaluations of interlaminar epidural steroid injections.

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